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Lower extremity muscle activation onset times during the transition from double-leg stance to single-leg stance in anterior cruciate ligament injured subjects

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ABSTRACT

The goal of this study was to evaluate muscle activation onset times (MAOT) of both legs during a transition task from double-leg stance (DLS) to single-leg stance (SLS) in anterior cruciate ligament injured (ACLI) ($n = 15$) and non-injured control subjects ($n = 15$) with eyes open and eyes closed. Significantly delayed MAOT were found in the ACLI group compared to the control group for vastus lateralis, vastus medialis obliquus, hamstrings medial, hamstrings lateral, tibialis anterior, peroneus longus and gastrocnemius in both vision conditions, for gluteus maximus and gluteus medius with eyes open and for tensor fascia latae with eyes closed. Within the ACLI group, delayed MAOT of tibialis anterior with eyes open and gastrocnemius with eyes closed were found in the injured leg compared to the non-injured leg. All other muscles were not significantly different between legs. In conclusion, the ACLI group showed delayed MAOT not only around the knee, but also at the hip and ankle muscles compared to the non-injured control group. No differences between both legs of the ACLI group were found, except for tibialis anterior and gastrocnemius. These findings indirectly support including central nervous system re-education training to target the underlying mechanisms of these altered MAOT after ACL injury.

1. Introduction

Tears of the anterior cruciate ligament (ACL) continue to be one of the most devastating sports injuries of the lower extremity, leading to a long and often difficult rehabilitation. The development of adequate dynamic knee joint stability after ACL injury is largely dependent on an optimal functioning of the neuromuscular system and is of crucial importance for rehabilitation (Williams, Chmielewski, Rudolph, Buchanan, & Snyder-Mackler, 2001). To optimize rehabilitation approaches

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and improve clinical outcomes after ACL injury, it is therefore essential to enhance our understanding of neuromuscular control deficits after ACL injury (Wikstrom, Tillman, Chmielewski, & Borsa, 2006; Williams et al., 2001).

Most of the previous literature measuring muscle activation patterns following ACL injury focused on the performance of muscles surrounding the injured knee joint (hamstrings, quadriceps and gastrocnemius) during a variety of experimental tasks (Ingersoll, Grindstaff, Pietrosimone, & Hart, 2008). An adaptive motor pattern that involves an increased hamstrings activity (Aalbersberg, Kingma, & van Dieen, 2009; Boerboom et al., 2001; Branch, Hunter, & Donath, 1989; Ciccotti, Kerlan, Perry, & Pink, 1994; Houck, Wilding, Gupta, De Haven, & Maloney, 2007; Swanik, Lephart, Giraldo, DeMont, & Fu, 1999; Swanik, Lephart, Swanik, Stone, & Fu, 2004) and decreased quadriceps activity (Branch et al., 1989; Gauffin & Tropp, 1992; Houck et al., 2007; Lynch, Logerstedt, Axe, & Snyder-Mackler, 2012; Swanik et al., 1999) has been observed in ACL injured (ACLI) subjects. This neuromuscular strategy may be the result of a series of complex neurophysiological alterations after ACL injury with the purpose to prevent excessive loads around the injured knee (Branch et al., 1989; Pietrosimone, McLeod, & Lepley, 2012; Swanik et al., 1999). However, a substantial variety between individual muscle activation patterns may exist, which can be related to the functional status after injury (Boerboom et al., 2001; Bryant, Newton, & Steele, 2009; Chmielewski, Rudolph, & Snyder-Mackler, 2002; Houck et al., 2007; Rudolph, Axe, Buchanan, Scholz, & Snyder-Mackler, 2001). Apart from the amount of activity of specific muscles, a precisely timed and coordinated muscle activation is also imperative to develop and maintain optimal joint stability during functional activities (Comerford & Mottram, 2001; Hewett, Zazulak, Myer, & Ford, 2005; Wikstrom et al., 2006) and avoid the development of maladaptive movement patterns which may predispose the ACLI subjects to an early onset of osteoarthritis and functional limitations (Kaplan, 2011; Trulsson, Miller, Hansson, Gummesson, & Garwicz, 2015), even when an ACL reconstruction is performed (Goerger et al., 2015). A delayed muscle activation of the hamstrings (Beard, Kyberd, Fergusson, & Dodd, 1993; Wojtys & Huston, 1994), quadriceps and gastrocnemius (Wojtys & Huston, 1994) in response to the application of an anterior tibial force was found in ACLI subjects. In contrast, other studies reported an earlier hamstring (Kalund, Sinkjaer, Arendt-Nielsen, & Simonsen, 1990; Sinkjaer & Arendt-Nielsen, 1991), quadriceps (Sinkjaer & Arendt-Nielsen, 1991) and gastrocnemius (Lindstrom, Fellander-Tsai, Wredmark, & Henriksson, 2009; Sinkjaer & Arendt-Nielsen, 1991) activation during walking after ACL injury, or no significant differences in timing of muscle activation during a single-leg hop for distance (Bryant et al., 2009). Klyne, Keays, Bullock-Saxton, and Newcombe (2012) reported a prolonged activity of medial gastrocnemius during a single-leg hop test in ACLI subjects. The variety of experimental tasks, methodologies and time since injury in these previous studies may have contributed to this lack of consistent evidence concerning the timing of muscle activation after ACL injury. Furthermore, the effects on muscle activation patterns of the non-injured leg remain largely unknown. Bilateral neuromuscular quadriceps deficits after unilateral ACL injury have been reported (Urbach, Nebelung, Weiler, & Awiszus, 1999), which may be attributed to alterations in the organization of the central nervous system (CNS) after ACL injury (Baumeister, Reinecke, Schubert, & Weiss, 2011; Baumeister, Reinecke, & Weiss, 2008; Grooms, Appelbaum, & Onate, 2015; Kapreli et al., 2009; Needle et al., 2014).

Muscle activation patterns around the proximal (hip) and distal (ankle) lower extremity joints after ACL injury are less studied, despite the suggestions that alterations in neuromuscular control may occur at different joints of the lower extremity after peripheral joint injury (Riemann & Lephart, 2002b). In subjects with chronic ankle instability for example, it has been shown that muscle activation onset times were delayed not only at the ankle but also at the hip and hamstring muscles during the transition from double-leg stance (DLS) to single-leg stance (SLS) compared to non-injured control subjects (Van Deun et al., 2007). During the transition from DLS to SLS, lower limb posture of the upcoming stance limb needs to be controlled by muscles functioning in the frontal and transversal plane, and not exclusively by muscles functioning in the sagittal plane, which is consistent with the multidirectional function of the ACL (Quatman et al., 2014). The stabilizing muscles of the hip, including gluteus maximus and gluteus medius, play an important role in controlling lower extremity and pelvic postures and movements during functional activities especially in the frontal and transversal plane (Ford et al., 2015; Willson, Ireland, & Davis, 2006; Zazulak et al., 2005), but the changes that may occur in these muscles following ACL injury remain unclear. With respect to the ankle muscles, an increase in the duration of tibialis anterior activity during gait (Lindstrom et al., 2009) and an increased tibialis anterior activity during a variety of functional activities (Ciccotti et al., 1994) were previously reported in ACLI subjects, possibly as a compensatory mechanism in the transversal plane by decreasing foot pronation and subsequent tibial internal rotation (Ciccotti et al., 1994).

Based on these earlier mentioned shortcomings and mixed findings in literature, there is a need to investigate neuromuscular control deficits within the lower extremity of both the injured and non-injured leg after ACL injury. Therefore, the goal of this study is to investigate lower extremity muscle activation onset times after ACL injury during a transition task from DLS to SLS during eyes open and eyes closed conditions. Our first hypothesis was that the ACLI group would show delayed muscle activation onset times compared to a non-injured control group, not only in muscles surrounding the knee, but also at the hip and ankle. Second, we hypothesized that these differences would become more apparent during the eyes closed condition, as subjects with an ACL injury may become more dependent on visual information to perform postural control tasks as a result of the altered proprioceptive information coming from the knee after ACL injury (Dingenen et al., 2015a; Grooms et al., 2015). Third, we hypothesized that no significant differences between the injured and non-injured leg of the ACLI group would be found, based on the earlier mentioned possible alterations in the CNS and the assumption that muscle activation patterns during voluntary postural control tasks are centrally mediated (Bouisset & Do, 2008).